EXERCISE IN THE PRACTICE OF MEDICINE

by

GERALD F. FLETCHER, M.D.

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DEDICATION

To my mother of 81 who has supported me in endeavors through the years; who, during the course of the final phase of preparation of this book, fell and fractured her left distal femur; but after surgical intervention combined with an optimistic attitude began active physical therapy early and is now easily moving about home displaying the effects of early activity and exercise despite a rather severe surgical disability.

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PREFACE

As the 1980s begin, physical activity and exercise are assuming an increasingly important role in the day-to-day life-style of the American public. This exercise enthusiasm has developed both in the allegedly healthy and unhealthy populations. Numerous programs for organized and medically supervised physical activity in cardiac patients (post infarction and post myocardial revascularization) exist over the country. These programs offer a multifactoral approach for the subject, including efforts to modify dietary habits and body weight, control blood pressure, eliminate smoking, and modify certain behavioral characteristics.

The history of physical activity relating to health and medicine is to some degree becoming cyclic or repetitive. Before the era of machines and conveniences of life, our population, by necessity, was much more involved in physical activity and physical labor. Then with the industrial revolution and the evolution of our "mechanized society," people became less physically active and the sedentary way of life became more "the rule than the exception." Of late (within the last ten years) as the public has become more "health conscious," there has been a resurgence of exercise and physical activity.

An historically important phase of exercise evolved in 1818 when William Cubitt, a British civil engineer, designed an elongated "stepping wheel" on which dozens of prisoners could work side by side. In accord with this, "treading the wheel" for punishment became prevalent throughout English prisons.[1] In 1846, however, because reformers considered treading the wheel a cruel, inhumane, and unhealthy practice, Edward Smith began to investigate physical performance by utilizing new physiologic techniques during treadmill exercise.[2] These studies were the first systematic inquiry into the respiratory and metabolic response of the human to muscular exercise, and provided the groundwork for extensive studies that have followed in the evaluation of exercise performance in patients with coronary heart disease. Smith's early studies included measurements of inspired air, respiratory rate, stroke

volume, pulse rate, and oxygen production. By 1857, measurements were made for other types of exercise including swimming, rowing, and horseback riding.[3]

With the acquisition of exercise habits early in history and the insight into methods of evaluation of exercise performance, observations[4] in some instances began to reveal a higher mortality rate in people with sedentary occupations compared to those who were physically active. These observations were to be the forerunners of more studies relating physical inactivity to mortality and morbidity from coronary heart disease.

Later in the 1900s Dr. Paul Dudley White became part of history and his influence will continue in both professional and lay circles. He was a strong advocate of exercise and believed that exercise was of value, both physiologically and psychologically. He felt that walking was beneficial, especially because of its "venous squeezing" effect, that the lower extremity veins have valves, and that walking helped move the blood in the veins and provoked better circulatory dynamics. He advocated that people who walk a great deal have less early arteriosclerosis and felt that people today have a life that is much too easy—using elevators instead of stairs and having lunch brought in rather than going out. He believed that "work alone never killed a man unless he was already sick."

Dr. White advised walking as probably the best exercise and was averse to activities with weight lifting. His own personal exercise included climbing stairs, walking to lunch, gardening, cutting trees, splitting logs, shoveling snow, and "working in the soil."

He believed that "one feels so much better with exercise" and his formula for long life was to "work hard mentally, physically, and spiritually." He referred to our easy way of living as a "real pity" and felt that our ancestors were in better physical health because of their active lives spent in clearing the forests and plowing the land. He believed that exercise was the "best tranquilizer there is." In farewell comments to his many friends and acquaintances, he was never known to say "take it easy" but rather to say "take it hard."[5]

With the current interest in exercise, physicians are repeatedly confronted with the problem of the proper prescription for the individual patient. In many instances there are considerations of numerous disease states. In all of these subsets of medical practice, physicians have increasing need to be familiar with the application of exercise testing and the exercise prescription within the safe limits for the individual's need and the residual disease involved.

Information to follow in this book will include a brief but in-depth coverage of Clinical Physiology by Dr. Robert Schlant of Emory Univer-

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sity School of Medicine, followed by a thorough but practical section on Exercise Testing by Dr. L. Thomas Sheffield of the University of Alabama School of Medicine. Emphasis will be placed on exercise in both the primary and secondary prevention of cardiovascular disease with precise guidance for the prescribing and programming of exercise in a section provided in collaboration with Dr. J. Ronald Mikolich.

The subspecialties of Internal Medicine will be considered with respect to exercise. These will be highlighted in a special section on Exercise and Pulmonary Disease by Dr. Jonne B. Walter of Georgia Baptist Medical Center. This will be followed by more concise sections on Exercise in Hypertensive and Peripheral Vascular Disease, Hematology, Neurology, and Endocrinology. Exercise will also be discussed as it relates to other major specialties of Medicine-Obstetrics and Gynecology, Pediatrics, Psychiatry, General Surgery, and Orthopedics. Data in these specialties are limited and it is the hope of the author that future publications will include more input from experts in these particular areas.

Special discussions will then follow on the Influence of Environmental Factors on Physical Activity and the Dangers of Exercise.

The author would like to express appreciation to the staff members of the Georgia Baptist Medical Center Cardiac Rehabilitation Program who have provided stimulation and information for the writing of this book. Dr. John D. Cantwell, who has interest both in preventive cardiovascular health and rehabilitation, is an ever present contributor of information and experience in this area. Barbara L. Johnston, MN (Nurse Coordinator of Cardiac Rehabilitation), has provided clinical research in many areas, especially with contributions on environmental factors related to physical activity. In the secretarial staff, Ms. Lillian McKinney as the manuscript typist has provided precise and patient attention to the meticulous preparation of the material. Ms. Barbara Chumbler, Ms. Debbie Guinn, and Ms. Patty Holloman have been ever present and loyal supporters of the endeavor.

As this book is finalized, new information is evolving. Certainly the major specialties of medicine and likely the subspecialties of internal medicine will become more involved with exercise both with regard to testing and preventive and therapeutic measures. It is our hope that this book will be only a brief and incomplete beginning of what the future holds.

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REFERENCES

1. Fletcher GF, Cantwell JD: Historical aspects of exercise and coronary heart disease. In: *Exercise and Coronary Heart Disease*. 2nd ed. Springfield, Illinois, Charles C. Thomas, 1979:7.
2. Chapman CB, Smith E (1818–1874): Physiologist, human ecologist reformer. *J Hist Med* 1967; 22:1–26.
3. Smith E: Inquiries into the quantity of air inspired throughout the day and night and under the influence of exercise, food, medicine and temperature. *Proc Roy Soc Med* 1957; 8:451–456.
4. Smith E: Report on the sanitary circumstances of tailors in London. In: *6th Edition Rep Med Officer Primary Council with Appendix*, 1862. London, H.M. Stationary Office 1864: 416–430.
5. White, PD: Personal communication, 1969.

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PHYSIOLOGY OF EXERCISE

Robert C. Schlant, M.D.

Exercise ferments the humors, casts them into their proper channels, throws off redundancies, and helps nature in those secret distributions, without which the body cannot subsist in its vigor, nor the soul act with cheerfulness.
Joseph Addison, 1711

INTRODUCTION

Exercise is one of the most common of all activities, and some form of exercise is performed by all normal individuals. On the other hand, the neural, metabolic, and physiologic adjustments and control systems that must be integrated during ordinary exercise are extraordinarily complex. The purpose of this chapter is to review briefly the physiology of exercise, more detailed reviews of which are available.[1–4]

TYPES OF EXERCISE

Exercise is usually divided into the following two types: (1) rhythmic or dynamic (isotonic) exercise is the usual, repetitive, rhythmic contraction of muscle groups associated with nearly continuous motion of part of the body. Examples include walking, running, and swimming. (2) Static or isometric exercise consists of sustained muscle contractions that produce no or relatively little motion of the involved body part. Examples include squeezing a hand dynamometer, carrying a heavy suitcase, and water skiing. Obviously, some forms of exercise involve both dynamic and static exercise. The following discussion will refer to dynamic exercise except when specifically stated otherwise.

PHASES OF EXERCISE

The cardiovascular responses to continued dynamic exercise can be divided into the following four phases: (1) the anticipatory or expectant phase, (2) the on-transient or initiation phase, (3) the adjustment or fine-tuning phase, and (4) the "drift" period.

Anticipatory Phase

This phase occurs immediately prior to the onset of exercise. The effects are produced by the autonomic nervous system and are under conscious and subconscious cerebral influences probably initiated from the cerebral cortex and diencephalon. It is associated with increases in heart rate (HR), blood pressure, and cardiac output (Q) in association with a decrease in venous compliance and an increase in venous return to the heart. Vasodilatation of the arterioles in the skeletal muscles may be produced by sympathetic cholinergic impulses.[5]

Initiation Phase

An intense increase in autonomic nervous system activity occurs almost immediately with the onset of exercise and produces further increases in heart rate, blood pressure, and cardiac output (Fig. 1).[6]

Figure 1. Cardiac output ($\dot{Q}$) in l/min and estimates of its regional distribution in relation to the oxygen uptake ($\dot{V}O_2$) at rest and during submaximal and maximal leg exercise (bicycling) and arm exercise (arm cranking). At any $\dot{V}O_2$, values apply to the situation after 5–7 minutes of exercise when $\dot{V}O_2$ and $\dot{Q}$ have reached a steady state. In the diagrams corresponding values are shown for heart rate (HR) in beats/min, for stroke volume (SV) in ml, and for aortic pressures (BP)— systolic, mean, and diastolic—in mmHg. Based on data from Clausen et al,[22] Rowell,[71] Kitamura et al,[306] and Wade Bishop.[12] It should be noted that during maximal exercise as well as at any relative $\dot{V}O_2$, the perfusion of non-working tissues and the heart can be assumed to be the same during arm exercise as during leg exercise and at any absolute $\dot{V}O_2$, $\dot{Q}$ is the same for the two types of exercise. MBF=Muscle Blood Flow; COR=Coronary Blood Flow; Skin=Skin Blood Flow; Viscera= Visceral Blood Flow; CBF=Cerebral Blood Flow; HR= Heart Rate; SV=Stroke Volume; BP=Blood Pressure. (From Clausen JP, Circulatory adjustments to dynamic exercise and effect of physical training in normal subjects and in patients with coronary artery disease. *Progress in Cardiovascular Disease* 1976; 18:459, by permission.)

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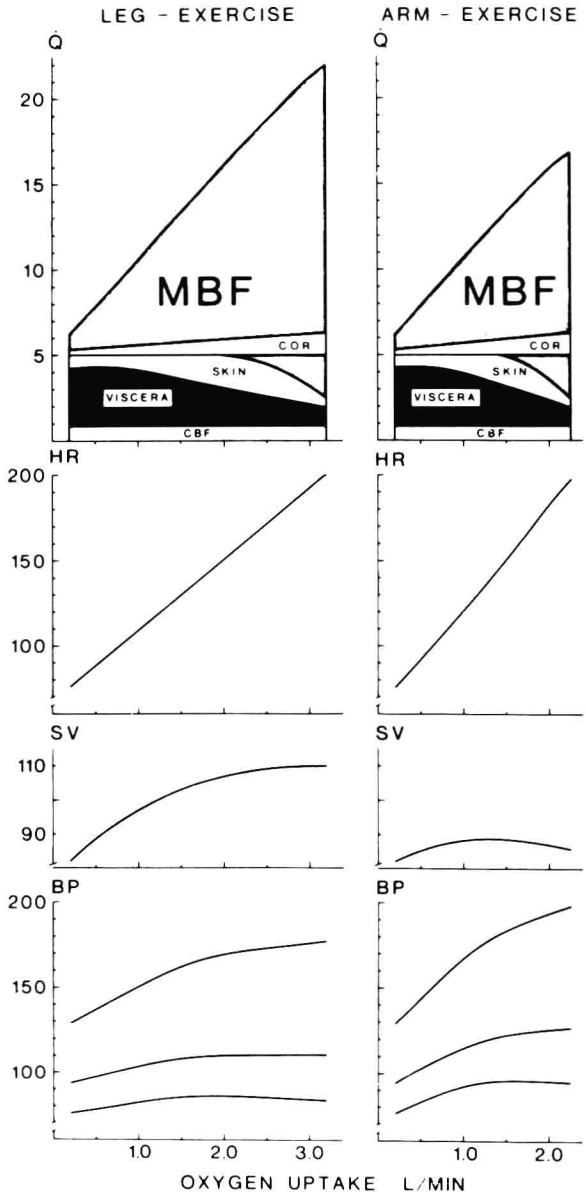
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100
1.0
2.0
3.0
OXYGEN UPTAKE L/MIN

Figure 1.

The heart rate increases rapidly, within seconds. The initial increase in heart rate is due to vagal withdrawal, whereas after 8–10 seconds, there is a large sympathetic accelerator component.[7–9] At times, the heart rate overshoots and then comes back down to a steady rate.[10] The increase in heart rate is a function of the relative intensity of the work.[11] The heart rate may increase 2½ to 4 times normal during very severe exercise;[12–15] in some highly trained individuals, the heart rate may increase from 50 to 220.[6,13,16,17] The increase in heart rate may also be partly due to the large increase in venous return activating the Bainbridge reflex and producing cardiac acceleration.[6,7,18] In addition, it appears likely that afferent impulses originating from the exercising muscles also contribute to the increased sympathetic stimulation of the heart and to the tachycardia during steady-state exercise.[19] In general, the heart rate is faster during arm cranking than during bicycling,[20–22] faster during bicycling than running,[23,24] and faster during running than walking with ski sticks ("ski-walking").[24]

The usual increase in cuff arterial blood pressure during exercise is due both to the increased cardiac output and to the sympathetic vasoconstriction of nonexercising muscles, viscera, and skin.[9,15,25,26] It appears that the arterial baroreceptor reflex is markedly inhibited during severe exercise.[27] It is important to note that studies have demonstrated that systolic blood pressure in the aorta during exercise increases much less than radial systolic blood pressure but that the increases in mean pressures were nearly identical.[28–31] Of interest, the same amount of upper arm exercise produces a greater increase in blood pressure than the same amount of work by the legs, apparently due to more intense vasoconstriction in nonexercising tissues during arm exercise (Fig. 1).[21,22]

The increase in cardiac output, which may reach a plateau in less than a minute,[32] goes mainly to the exercising muscles and to the myocardium (Fig. 1).[33,34] In general, the increase in cardiac output is nearly proportional to the increase in total oxygen consumption, which can increase from 250 ml/min (or 3.5 to 4.5 ml O_2/kg/min) to 6.0 liters per minute (or 75 to 80 ml O_2/kg/min) in physically trained men (Fig. 2).[4,35,36] The increase in cardiac output is the combined result of an increased venous return produced by mechanical compression ("milking") of the veins in the exercising muscles, compression of the intra-abdominal veins by the abdominal wall, and the action of the thoracic "respiratory pump";[37–40] an increase in heart rate; an increase in myocardial contractility (and the rate of relaxation) from the positive

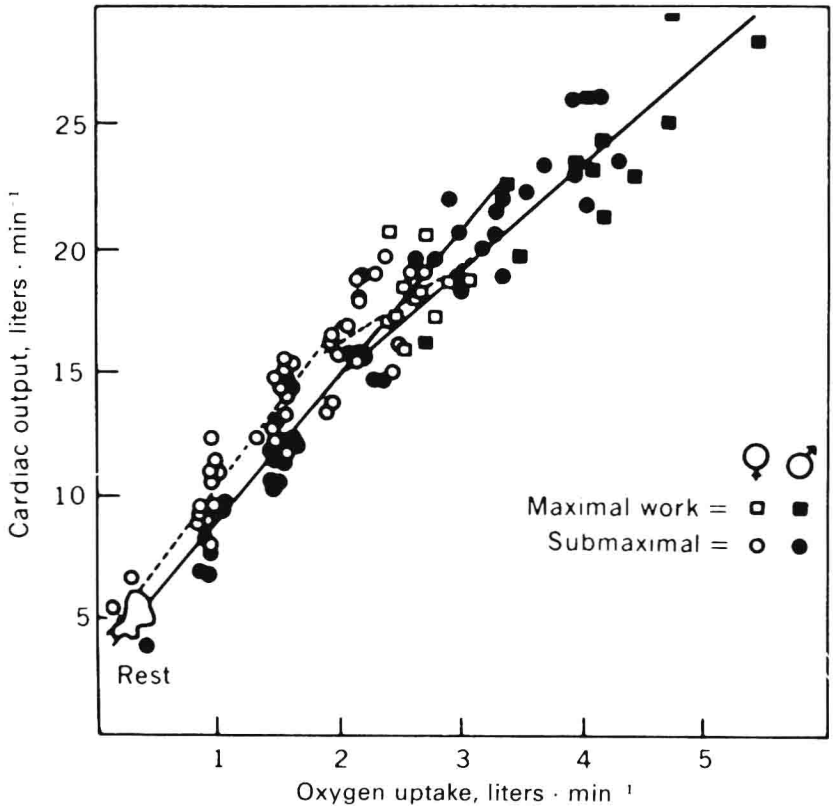

Figure 2. Individual values on cardiac output in relation to oxygen uptake at rest, during submaximal, and during maximal exercise on 23 subjects sitting on a bicycle ergometer. Regression lines (broken lines for women) were calculated for experiments where the oxygen uptake was (1) below 70 percent and (2) above 70 percent of the individual's maximum. (From *Textbook of Work Physiology*, 2nd Edition, by Astrand P-O, Rodahl K, copyright 1977 by McGraw-Hill Book Company, Inc. Used with the permission of McGraw-Hill Book Company.)

inotropic sympathetic impulses to the heart and from the increase in heart rate (Treppe effect); and, especially, an acute decrease in afterload from the marked decrease in the peripheral resistance of the exercising muscles.[1,4,15,32,40–46] In some highly trained athletes, the cardiac output may increase five to six times the normal value, or from 5.0 liters per minute to 35–42 liters per minute. In general, the cardiac output at a given oxygen uptake during submaximal exercise in the upright posi-

tion is 1–2 liters less than during exercise in the supine position.[12,47,48]

Changes in stroke volume during exercise depend upon whether the exercise is performed in the supine position or in the upright position.[23,49–53] In the supine position, the stroke volume and end-diastolic volume are at a near maximal level at rest and thus do not increase or only increase relatively little, perhaps 10 to 20 percent, during supine exercise.[54] When one stands upright, however, stroke volume may decrease by 40 percent in association with a shift of 300–800 ml blood to the legs (Fig. 3).[55–57] During mild upright exercise, the increase in heart rate may be sufficient to produce all of the required increase in cardiac output.[58,59] Soon after the onset of vigorous upright exercise, however, stroke volume begins to increase, and during severe exercise, it may increase up to twice the resting value (Fig. 3).[9,14,22,23,26,51,53,57,60–62] Cardiovascular endurance training increases the elevation in stroke volume during exercise;[13] this increase in stroke volume during exercise is one of the better indicators of physical training. As an example, a highly trained athlete may increase his stroke volume from 85 ml to 170 ml. In general, stroke volume is greater during leg exercise than during arm exercise at all absolute and relative work levels.[9,21,22]

During moderately severe upright exercise, the left ventricular end-diastolic volume normally does not increase,[6,57,63–65] and any increase in stroke volume is produced by a decrease in end-systolic volume secondary to the combined effects of the increased contractility from beta-adrenergic stimulation and the decrease in afterload, rather than by an increase in end-diastolic volume or preload. Thus, in human beings at least, the Frank-Starling mechanism appears to play only a slight role in the normal ventricular response to exercise.[6] With extreme exercise, however, healthy dogs have been shown to have both a decrease in ventricular end-systolic volume and an increase in end-diastolic volume.[14,16] With most forms of exercise, the right atrial pressure decreases slightly, although it may increase slightly with very severe exercise.[15]

The vascular resistance of working muscles decreases within 0.5 seconds after the onset of exercise, almost instantaneously with muscle contraction.[1,15,32,41–44,46,66] When many muscles are used, therefore, the total systemic (peripheral) resistance of exercising muscles appears to be largely of local metabolic origin, perhaps mediated by ATP or adenosine.[42,44,45,67] Blood flow to the exercising muscles is also significantly aided by the rhythmic pumping effect of the muscles upon

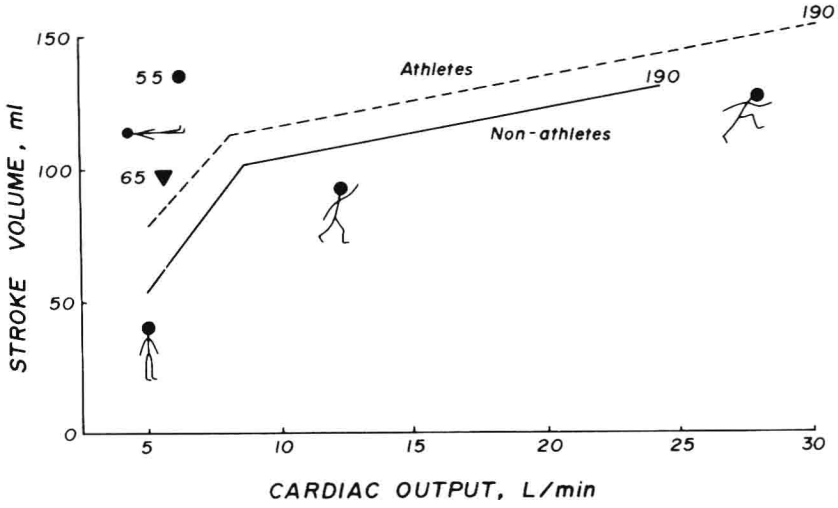

Figure 3. Cardiac output and stroke volume in athletes (●) and nonathletes (▲), during resting supine, quiet standing, and graded upright exercise. Figures represent the heart rate (beats/minute). The pattern in both groups is the same, except the athletes have a larger stroke volume and a slower heart rate when resting. On standing, the stroke volume decreases because of pooling of blood in the lower limbs. Even with mild exercise the muscle pump restores the pooled blood to the heart and lungs, and the stroke volume approaches the supine value. As exercise increases in severity, the stroke volume continues to increase slightly, so that as maximal exercise is approached it exceeds by about 10 percent the values obtained in the supine position at rest. Oxygen consumption in both groups at rest is about 300 ml/min, and at maximal effort it is about 3,000 ml/min in the nonathletes and about 5,000 ml/min in the athletes. (Shepherd JT, VanHoutte PM: *The Human Cardiovascular System*, 1979, reprinted by permission of Raven Press, New York.)

the arteries and veins, which increase local perfusion pressures and increase muscle oxygenation.[39,46] The intense sympathetic adrenergic outflow to the splanchnic area and to nonexercising muscles produces both arterial vasoconstriction, which decreases blood flow to the nonexercising muscles, skin, kidney, spleen, liver, and intestines, and venoconstriction, which increases venous return from the splanchnic capacitance vessels.[9,12,15,22,46,68–71] Reduction in splanchnic-hepatic-renal blood flow produced by sympathetic vasoconstriction is a function of the relative work load and is similar to the increase in heart rate.[22,71] In contrast to most studies in human beings, however, studies in exer-

cising dogs have indicated that visceral flow is relatively well maintained so long as all other compensatory mechanisms remain intact, indicating that the normal dog uses his sympathetic system less than man under stress.[6,33,34,71–78] Cerebral blood flow (CBF) in man probably stays the same or increases slightly during exercise,[79] while coronary blood flow normally increases markedly during sustained exercise.[9,80–84]

Exercising muscles may extract twenty times more oxygen than usual, producing a widened arteriovenous oxygen difference together with increased K^+ in the venous blood and increased tissue osmolality.[85–88] The arteriovenous oxygen difference of the body can increase approximately 2.5 to 3.0 times its resting value, or from 5 to 15 ml oxygen per 100 ml blood. In addition, during heavy exercise, exercising skeletal muscles utilize some "anaerobic" metabolism and release lactate. Working muscles may develop a temperature above 40°C and a pH lower than 7.0 from the formation of CO_2 and lactic acid.[87–89] The increase in body temperature and lower pH may produce a "Bohr effect" on the oxygen-hemoglobin dissociation curve, helping to deliver more oxygen to the working muscles. On the other hand, arterial saturation may decrease slightly during strenuous exertion due to the same Bohr effect in the pulmonary capillaries.[4]

At low levels of exercise and low levels of force development, skeletal muscles appear to use predominately "red" slow twitch (ST) or Type I muscle fibers, which are rich in mitochondria and intramitochondrial enzymes necessary for the citric acid cycle, the fatty acid cycle, and the electron transport chain.[90] Such Type I fibers can sustain rhythmic contraction for long periods of time. When greater force is necessary, the muscle units appear to recruit "white" fast twitch (FTb) or Type IIb muscle fibers, which have fewer mitochondria but have a high content of enzymes for anaerobic glycosis. Some of the progressive release of lactate during heavy work loads is probably explained by the recruitment of Type IIb muscle fibers rather than by hypoxia.[9,91]

Untrained individuals may develop pain or a "stitch" in the side during this or a later phase, presumably from hypoxia of the diaphragm resulting from increased respiratory work prior to the redistribution of blood flow.

Adjustment Phase

During this phase, there is a complex integration of central and peripheral mechanisms to maintain cardiac output, venous return, and adequate distribution of the cardiac output to the working muscles. The

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